Chapter 9

Metabolic Disorders

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I. METABOLIC DISORDERS OF RUMINANTS

A. Parturient paresis (hypocalcemia, milk fever)

1. Patient profile and history
   a. Parturient paresis is most commonly seen in dairy cattle and may affect 5%–10% of all adult dairy cows. This disease is less common in beef cattle, sheep, and goats. There is a breed susceptibility, with Jerseys and Guernseys having a higher incidence of disease than other dairy breeds.
   b. Usually, there is a distribution of cases around parturition, with 75% of clinical cases occurring within 24 hours of calving, **12%** occurring **24–48** hours after calving, and 6% occurring at calving. Hypocalcemia at calving often is associated with dystocia. A subset (7%) also occurs before or unassociated with calving. These forms of the disease are associated with calcium loss but are not caused by an onset of lactation.
   c. There is an age susceptibility to the disease in that it parallels milk production, and it is rare in heifers. There is an individual susceptibility and the tendency for the condition to reoccur in susceptible cows.
2. Clinical findings
   a. Cattle. This condition is divided into three stages:
      (1) Stage **I.** The signs consist of mild excitement and tetany without recumbency. Anorexia is also a consistent finding. These signs may go unobserved because stage I rapidly progresses to stage II (1-hour progression).
      (2) Stage **II**
         (a) There is depression, paralysis, and recumbency. The head is characteristically turned into the flank or rested on the ground in an extended position. Fine muscle tremors may be evident, and the cow may make threatening motions with the head (e.g., head shaking, open-mouth bellowing).
         (b) Examination reveals tachycardia with decreased heart sounds, cool extremities, and a low rectal temperature (35.5°C–37.8°C). Gastrointestinal atony (e.g., mild bloat, constipation), loss of anal reflex, and a slow pupillary light reflex are evident. The clinical signs at this stage may be associated with uterine prolapse. Stage II may last from 1 to 12 hours.
      (3) Stage **III.** Cattle with stage III milk fever exhibit further weakness and progressive loss of consciousness. Bloat may be life threatening because of lateral recumbency and gastrointestinal tract (GIT) atony. There is a danger of aspiration pneumonia, the heart sounds become inaudible, and a pulse may be undetectable. This stage may progress to death in **3–4** hours.
   b. Sheep are more likely to develop milk fever in late gestation. Ewes usually exhibit flaccid paralysis; however, tetany or muscle tremors may be evident. Recumbency is less common than with cattle.
   c. Coats. Milk fever may occur prepartum, as in sheep, or postpartum, as in high-producing dairy animals. The clinical signs are similar to those in sheep.
3. Etiology and pathogenesis
   a. Hypocalcemia. When calcium outflow is sudden and severe (i.e., lactation in cows, multiple fetuses in sheep), calcium homeostasis mechanisms may fail. Mature cows have increased lactation demands for calcium and older cows have decreased ability to mobilize calcium from the GIT. GIT stasis at parturition (or at other times) also interferes with calcium uptake. Paralysis occurs because of inadequate calcium availability at the neuromuscular junction. Calcium is necessary for the release of acetylcholine. Also, low calcium levels impair muscular

contractility by hindering calcium-dependent actin and myosin interactions. Neuromuscular dysfunction may be variable (tetany or flaccid paralysis) with mildly decreased calcium concentrations or in nonbovine species (i.e., sheep, goats, horses). Retained placentae are common sequelae to milk fevers.

b. Hypophosphatemia. Simultaneous with calcium loss, phosphorus may be lost in milk or not absorbed (decreased intake or decreased mobilization from bone), causing the hypophosphatemia that is seen. Hypophosphatemia may result in enhanced flaccid paralysis and continued recumbency even after low serum calcium levels have been corrected.

c. Hypermagnesemia is seen occasionally and also enhances flaccidity. Increased levels of magnesium impair acetylcholine release. The cause for hypermagnesemia is unknown.

4. Diagnostic plan. The field diagnosis is based on clinical signs, history, and response to therapy. At the time of treatment, serum should be drawn and held for calcium analysis.

5. Laboratory tests

a. Total serum calcium determination is the standard test for all ruminants. Although ionized calcium is the active form, it is difficult to measure in most laboratories and usually closely parallels total calcium concentrations. Total calcium less than 1.9 mmol/L may result in clinical signs, with stage I disease usually characterized by a serum calcium level of 1.4–2.0 mmol/L. Most animals become recumbent before a level of 1.25 mmol/L is reached (the typical range of stage II milk fever is 0.9–1.6 mmol/L). With stage III disease, levels may reach 0.5 mmol/L before the animal dies.

b. Serum magnesium and phosphorus determination

(1) In dairy cattle with parturient paresis, hypermagnesemia (1.5–2 mmol/L) and hypophosphatemia (0.5–1 mmol/L) may be seen.

(2) In the less common presentation of nonparturient paresis (i.e., calcium loss not associated with calving), hyperphosphatemia and hypomagnesemia may occur.

6. Differential diagnoses include other causes of recumbency: trauma, peripheral nerve paralysis, calving injuries, or septicemias and toxemias (e.g., acute coliform mastitis).

7. Therapeutic plan. Owners may be taught how to diagnose and treat parturient paresis on their own.

a. **Cattle.** The disease should be treated as an emergency, particularly if the animal is in stage III. Prolonged recumbency results in complications (e.g., bloat, uterine prolapse, muscle necrosis, aspiration pneumonia). Animals do not recover spontaneously. Individuals respond best in early stages and with early treatment. Between 5% and 15% of cows do not respond to initial treatment and remain recumbent for various lengths of time. Persistent hypophosphatemia may be an underlying cause for failure to respond.

(1) Relieve bloat. Roll the cow into sternal recumbency before treatment to relieve any bloat.

(2) Administer calcium. Intravenous and subcutaneous calcium solutions each contain between 8 g and 10 g of calcium and are advocated in tandem to lessen the chance of relapse. One 500-ml bottle is sufficient for treatment, but calcium continues to be lost in the milk and is not being absorbed because of GIT atony and increased calcitonin release.

(a) Administer 500 ml of 23% calcium gluconate intravenously (calcium borogluconate and glucose, phosphorus, magnesium, and potassium are often found in commercial preparations). Intravenous injections must be given slowly (over 20 minutes) and the heart monitored for signs of irregularity. The heart rate should slow dramatically and become stronger over the period of calcium administration. Other responses to therapy include eructation, urination, defecation, muscle twitching, and an improvement in demeanor. Within 30 minutes of treatment, 60% of animals with uncomplicated milk fever will stand.

(b) Subcutaneous calcium. A subcutaneous calcium preparation without glucose (one 500-ml bottle) is administered as well. For stage I conditions, subcutaneous administration alone may be sufficient. Also, subcutaneous administration without intravenous calcium treatment should be considered in cases complicated by toxemia and shock due to cardiovascular compromise.

(3) Follow-up

(a) Full restoration of calcium homeostasis does not occur for 2–3 days. The owner should request a revisit if there has been no response to therapy within 12 hours of treatment. Retreatment may be warranted up to two more treatments.

(b) If the animal is unresponsive to therapy shortly after administration, prop her up in sternal recumbency, and provide secure footing in case the cow attempts to rise. Provide shelter and easy access to feed and water if recumbency is prolonged.

(c) Good nursing care (i.e., deep bedding, rolling from side to side 4–5 times per day) may be necessary to prevent muscle damage. Mechanical hip-lifting devices may encourage the cow to stand but should be used with great care and discretion to avoid muscle damage. More efficacious *lifters* include harnesses and inflatable mattresses.

(d) Consider only partial milking for 1–2 milkings post treatment, unless there is concurrent mastitis. Relapses of clinical hypocalcemia may occur, and decreasing the milk drain of calcium is thought to lessen the likelihood of relapse.

b. Sheep and goats. Calcium gluconate should be administered at 50–100 ml of a 23% solution intravenously. Consider cesarian sections for sheep if near term. Sheep developing hypocalcemia in late gestation often relapse.

8. Prevention

a. Dry cow diets, which are low in calcium content (i.e., nonlegume roughage), are preventive. Diets high in anions (chloride ions and sulfur compounds) compared to cations (sodium and potassium ions) tend to prevent hypocalcemia.

b. Vitamin D_3 preparations administered prepartum may prevent or ameliorate the clinical signs, but the timing of administration is critical.

c. Other preventives include culling cattle prone to the condition, keeping cattle on feed at the time of parturition (i.e., reduce episodes of indigestion), and administering oral calcium in the form of calcium gel at calving.

B. Downer cow syndrome

1. Patient profile and history. Cows that remain recumbent 24 hours after initially going down are classified as downer cows. The condition may or may not be related to milk fever, but the vast majority of cases are periparturient and related to parturient paresis.

2. Clinical findings. The animal does not appear to be systemically ill but either is unable to rise or refuses to rise. The cow may have been treated with calcium for parturient paresis. She may pull herself around her enclosure, which accounts for the term "creeper cow."

3. Etiology and pathogenesis. Causes of recumbency include parturient paresis, calving paralysis (obturator or sciatic paralysis), pelvic fracture, coxofemoral luxation, lymphosarcoma, and malnutrition. Any large animal that is recumbent is vulnerable to muscle damage. Compressive effects are evident within 6–12 hours on a hard surface. This damage is almost exclusively confined to muscles of the pelvic limbs.

4. Diagnostic plan. Eliminate any obvious causes of recumbency via a general physical examination, and perform a rectal and manual examination of the pelvis. The clinical examination may be aided by the use of a hip lifter or slings. However, the downer animal is often labeled as such by the elimination or exclusion of obvious causes of the condition.

5. Laboratory tests. Muscle leakage enzyme (creatine kinase) values rise to moderately high levels but fall to more normal levels after 6 days of recumbency. This may help to differentiate recumbency from other causes from a primary muscular disorder. Serum levels of calcium and phosphorus may be low if the primary disorder is caused by hypocaicemia.

6. Therapeutic plan
   a. Underlying conditions should be treated, if possible.
   b. Supplementation with phosphorus may be efficacious.
   c. Lifting devices, such as hip lifters, are useful but only for short periods (e.g., examination, milking). The cow must not be allowed to hang unattended for long periods of time. Abdominal slings are useful but must be well designed. Air bags seem most humane but cause respiratory embarrassment and cows may roll off. Water stalls or pools hold the best theoretical promise but are impractical in field situations.
   d. Nursing care is necessary for all down animals and includes rolling, providing deep bedding, cleaning wet skin, feeding, milking, watering, and other supportive measures.
   e. **Analgesics** (e.g., aspirin, phenylbutazone, flunixin meglumine) may be indicated to relieve muscle pain.

7. Prognosis. The prognosis for each case depends on the primary disorder and any secondary complications. Any prognosis is extremely difficult to offer if the animal is a true downer without evidence of another or primary disorder.

8. Prevention
   a. To prevent calving injuries leading to recumbency, provide calving pens for animals rather than calving in stanchions, make sure heifers are large enough at the time of parturition, and provide breeding advice on sire size.
   b. Prevent parturient paresis as described in I A 8. Treat recumbent animals vigorously.
   c. Provide resilient matting or deep bedding in stanchion or comfort stalls.

C. Postparturient hemoglobinuria

1. Patient profile and history. Postparturient hemoglobinuria is a disease of mature, high-producing dairy cattle. It is of relatively low incidence, sporadic, and seen in cows 2–4 weeks post calving. It is most commonly seen on particular farms in association with certain diets.

2. Clinical findings
   a. There is a precipitous drop in milk yield with sudden weakness (e.g., staggering) and anorexia. The urine is reddish-brown to black (hemoglobinuria), and there are signs of anemia (e.g., pale mucous membranes, tachycardia, an increased cardiac impulse). Jaundice develops terminally (3–5 days) with weakness, recumbency, and death.
   b. Signs of phosphorus deficiency may be evident in the herd, for example pica, infertility, and poor growth of calves with musculoskeletal disorders (osteodystrophy).

3. Etiology and pathogenesis
   a. This condition is associated with hypophosphatemia because of low dietary phosphorus intake. It is seen in nonsupplemented cattle grazing on phosphorus-deficient soils. Lactation drain exacerbates phosphorus loss. There may be an association with low total body copper and selenium in individual animals.
   b. It is felt that ingested **hemolytic** agents, such as cruciferous plants (kale and other brassicas), cause **erythrolysis,** particularly in cases of increased red blood cell fragility. Red blood cells are more fragile with mineral deficiencies (e.g., phosphorus, copper, selenium). The best documented mineral deficiency is chronic phosphorus deficiency.

4. Diagnostic plan. The field diagnosis is based on clinical findings. The clinician must differentiate other causes of red urine.

5. Laboratory tests
   a. Serum phosphorus. Low serum phosphorus levels are found in lactating, unaffected animals (0.65–1.0 mmol/L). Clinically affected cattle have very low serum phosphorus values (less than 0.5 mmol/L).
   b. Complete blood cell count (CBC) and hematocrit. Cattle are anemic as evidenced by the low total red blood cell count and hemoglobin.
   c. Urinalysis. There is hemoglobinuria.
   d. Serum copper levels may be low.

6. Therapeutic plan
   a. A blood transfusion (5–10 L/cow) must be administered to save the affected animal.
   b. Isotonic fluid therapy is necessary to provide diuresis and renal support.
   c. Phosphorus should be given intravenously at a dose of 60 g of sodium acid phosphate in 300 ml of distilled water with a similar amount subcutaneously. The subcutaneous treatment should be repeated once per day for the next 3 days coupled with the same daily dose orally.
   d. Dietary supplementation with bone meal at 120 g twice daily or some other source of phosphorus is recommended for 5 days post treatment.

7. Prognosis. The prognosis for any anemic recumbent animal is poor. Kidney failure may occur within 3–5 days, and if an animal survives, a long convalescence may be necessary.

8. Prevention. Phosphorus, copper, and/or selenium supplementation for the herd is warranted.

D. Clinical bovine ketosis (acetonemia)

1. Patient profile and history
   a. Primary ketosis is a metabolic disorder that occurs in lactating dairy cows 1–6 weeks after calving (usually 3–4 weeks into lactation). It may be seen in high-producing, heavily fed dairy cows indoors, and is rarely seen in cattle on pasture.
   b. Secondary ketosis may occur as a complication of another disease that usually occurs early in the postpartum period (e.g., metritis, left displacement of the abomasum).

2. Clinical findings
   a. Primary ketosis. Cows exhibit a gradual decrease in appetite and milk yield for 3–4 days. There is moderate weight loss and depression. Cows are selectively anorexic, preferring hay to grain or silage. Feces are characteristically firm and dry ("bull-like"). When appetite is lost, weight loss may be rapid. A ketone smell may be detectable on the cow's breath. Spontaneous recovery may occur when milk production and caloric intake are stabilized.
   b. **Nervous** ketosis is a type of primary ketosis where nervous signs predominate. The cow may appear delirious, walking in circles and head pressing with compulsive licking and chewing movements, hyperesthesia, bellowing, apparent blindness, and a depraved appetite.
   c. Secondary ketosis. Clinical signs are similar to those of primary ketosis but not as dramatic. The condition is accompanied by another clinical problem (e.g., metritis, left abomasal displacement). Signs may occur earlier in lactation than with primary ketosis (i.e., usually within 1–2 weeks of calving).

3. Etiology and pathogenesis
   a. Primary ketosis. Subclinical bovine ketosis may be normal in high-producing dairy cattle where there is often a negative energy balance early in lactation. High milk production causes energy (glucose) drain, and the need for energy may exceed capacity for intake. Likewise, the need for energy may exceed that provided for in the ration. A defect in digestion or metabolism may result in inadequate amounts of glucose available at the cellular level.
      (1) Hypoglycemia results in an increased requirement for gluconeogenesis in the liver (glucose from amino acids and glycerol). This pathway for glucose pro-

duction normally supplies a high percentage of glucose via the tricarboxylic acid (TCA) cycle. Interference with this pathway (e.g., lack of oxaloacetate) converts ketogenic volatile fatty acids to ketone bodies (acetoacetic and β-hydroxybutyric acid). Depletion of hepatic glycogen occurs and hepatic stores of triglycerides and ketone bodies increase.

(2) Ketogenic diets are thought to be low in precursors of propionic acid, which is converted directly to oxaloacetate and glucose. Ketogenic diets (e.g., silages) have high levels of butyric and acetic acid. High-protein diets also may be ketogenic in nature.

b. Secondary ketosis is thought to follow the same pathophysiologic sequence as primary ketosis; however, a causative or developmental factor is involved (e.g., a clinical disease). The clinical disease results in a decreased appetite, which, in the presence of the loss of lactose due to lactation, causes the ketotic syndrome to develop.

4. Diagnostic plan. The field diagnosis is based on history, clinical findings, cowside tests, and response to therapy. Laboratory tests confirm the diagnosis.

5. Laboratory tests
   a. With primary ketosis, blood glucose is depressed and blood, urinary, and milk ketones are elevated. A CBC reveals lymphocytosis, neutropenia, and eosinophilia. Cowside tests (i.e., dipstick evaluation) will detect urine and milk ketones.
   b. Secondary ketosis. Laboratory changes are similar to those of primary ketosis, but they are less dramatic.

6. Differential diagnoses include indigestion, traumatic reticuloperitonitis, abomasal displacements, and metritis. Central nervous system (CNS) diseases also should be considered when presented with a case of suspected nervous ketosis.

7. Therapeutic plan
   a. Routine therapy is the administration of *500* ml of 50% dextrose solution intravenously. Relapses are common and follow-up includes the oral administration of propylene glycol (225 g twice daily for 2 days followed by 110 g once daily for 2 more days).
   b. Corticosteroids (10 mg dexamethasone intramuscularly given once) may be considered for their effect on repartitioning glucose and depressing milk production. Anabolic steroids (60–120 mg of trenbolone acetate) are also efficacious in treatment. Be aware that this is an off label use in most countries.
   c. Vitamin B_{12} and cobalt may be administered, as may nicotinic acid, in the feed.

8. Prevention
   a. For herd problems, regularly check for ketonuria during the second to sixth weeks of lactation. Reduce the silage component of ration if practical. Sodium propionate or propylene glycol may be fed preventively, although this may be expensive.
   b. Cows should be in good condition at calving (not thin or overly fat). Dry off cows in good condition. Gradually increase the grain ration near the end of the dry period, and continue to increase feed after calving relative to milk production.
   c. The most important recommendations center on nutrition and dry cow management. Dividing the herd into separate feeding groups according to metabolic needs and production indices is necessary. Milking cows should receive good quality feed (16%–18% protein). Cows should receive adequate exercise during any stabling periods.

E. Subclinical bovine ketosis

1. Patient profile and history. Many high-producing dairy cattle in early lactation are subclinically ketotic. Affected animals are usually housed cattle. Subclinical ketosis can occur at a prevalence rate of 10%–30% in certain herds.

2. Clinical findings. Cows suffer from lower than expected milk production and gradual weight loss. There may be an infertility problem (e.g., metritis, ovarian dysfunction).

3. Etiology and pathogenesis. Causes are similar to those of clinical ketosis. Cows in negative energy balance with high glucose loss in milk (high milk production) and insufficient capacity for consumption or improper nutrition will be subclinically ketotic. Hepatic stores of glycogen decrease, and triglycerides and ketone bodies increase.

4. Diagnostic plan. Consider subclinical ketosis when there is a mild ketonuria, slight fall in milk production, and weight loss in an individual or across the herd. Clinical diseases causing secondary ketosis should be ruled out.

5. Laboratory tests confirm ketonuria and hypoglycemia.

6. Therapeutic plan. Recommendations are the same as for clinical ketosis (see I D 7).

F. Pregnancy toxemia of sheep

1. Patient profile and history. Risk factors for ewes developing the condition include late pregnancy, obesity, and carrying multiple (or single large) fetuses. This condition affects intensively raised sheep (i.e., not range sheep) and may be associated with sudden feed changes or inclement weather conditions. This may occur as a flock problem with several animals affected over several weeks.

2. Clinical findings
   a. Neurologic signs are similar to the signs of nervous ketosis in cattle. There is blindness and changes in demeanor. Animals may head press and circle or stand with a "star-gazing" appearance. Tremors and convulsions may occur interspersed by periods of depression, incoordination, and ataxia.
   b. Other signs. Ewes may be constipated and exhibit bruxism and a ketone smell to the breath. Terminally, ewes become recumbent and comatose, resulting in death in 4–7 days. Dystocia may be evident if ewes are in the process of lambing. Death of the ewe may be caused by toxemia resulting from death and decomposition of the fetuses.

3. Etiology and pathogenesis
   a. A period of anorexia or starvation (possibly resulting from feed change) for 1–2 days is the precipitating cause and may have been preceded by a gradual fall in the plane of nutrition during pregnancy. This disorder is not one of undernourished animals, but more often those in good flesh (over fit) with a recent decrease in consumption.
   b. The anorexia and starvation results in a hypoglycemia and hyperketonemia similar to bovine ketosis. The decrease in consumption is paralleled by a high-glucose drain from a single large fetus or multiple fetuses. The clinical picture is believed to be produced by a hypoglycemic encephalopathy. A terminal uremia may develop and exacerbate the condition.

4. Diagnostic plan. The diagnosis is made on the basis of clinical findings and history. Laboratory examination confirms the diagnosis. Collect urine by holding off the nares for a brief period, collecting a free-flow urine sample, and examining for ketone bodies.

5. Laboratory tests reveal hypoglycemia, ketonuria, eosinophilia, lymphocytosis, and neutropenia. Plasma cortisol levels are increased.

6. Differential diagnoses include hypocalcemia and CNS diseases (e.g., lead poisoning, polio, rabies).

7. Therapeutic plan
   a. A cesarian section is necessary immediately. Response to therapy without a cesarian is poor, and even with surgery, response is variable to poor.
   b. Clinically affected animals should be treated with intravenous glucose solutions and oral propylene glycol. Corticosteroids may be administered for their gluconeogenic effects.
   c. Follow-up. For recovering animals, force feed a highly palatable, readily digestible ration.

8. Prevention
   a. The rest of the flock should be examined daily for early clinical signs. Animals suspected of developing pregnancy toxemia should be treated orally with propylene glycol. Carbohydrate intake should be increased by providing supplemental feeding.
   b. A rising plane of nutrition should be ensured during the last 2 months of gestation. This may mean restricting feed intake in early gestation to avoid overly fat ewes at parturition. Avoid sudden feed changes during gestation and provide shelter and extra feed during cold and wet periods. Exercise is recommended for confined flocks.

G. Fat cow syndrome

1. Patient profile and history. This is a disease of overconditioned beef and dairy cattle. It may be an individual cow disease or a herd problem. There is a history of heavy feeding or a sudden decrease in nutrition in overly fat animals. The disease is sporadic but carries a high mortality rate.
   a. Dairy cows. Fat cow syndrome occurs in the immediate postpartum period, often concurrent with common postpartum diseases (e.g., metritis, retained placenta, displaced abomasum). There may be a history of a prolonged lactation or a dry period.
   b. Beef cows. The condition is most common in late gestation.
2. Clinical findings
   a. Individual
      (1) If an individual cow is affected, it is usually in an intensively managed situation, as with dairy cows. The animal is noticeably fat. The reason for examination may be for a usually responsive postpartum condition that is refractory to therapy. Anorexia and depression are pronounced. Ketosis is commonly severe, and response to therapy is poor and protracted.
      (2) If the cow continues to deteriorate despite therapy, CNS signs of cortical stimulation develop. Eventually, the animal becomes moribund.
   b. Herd
      (1) In dairy cows, there may be a high prevalence of unresponsive postpartum diseases (e.g., milk fever, ketosis, retained placenta, metritis). Failure of cows to become pregnant is a common complaint
      (2) With beef cattle in late gestation, the signs may be similar to pregnancy toxemia of sheep. Cattle appear nervous, excitable, and uncoordinated. Feces are firm and scant. Signs progress to recumbency, coma, and death.
3. Etiology and pathogenesis
   a. **Overfeeding** results in deposition of fat in body stores. Conditions occurring in late gestation (e.g., negative energy balance, hypoglycemia, high concentrations of lipolytic hormones, other poorly understood factors) stimulate mobilization of body fat. This results in increased uptake of fatty acids (FAs) by the liver.
   b. Fatty acids are a normal finding in all postpartum dairy cows as milk production outstrips digestive capacity. Usually, these FAs are esterified to triglycerides in the liver. Under normal circumstances, triglycerides are packaged into lipoproteins and transported to tissues for a source of energy or to the mammary gland for milk fat synthesis. Ruminant fat cow syndrome seems to occur when serum FAs are increased and triglycerides accumulate in the liver, while hepatic lipoprotein production does not increase or is reduced. It is suggested that fat begins to accumulate in the liver in late gestation, is dynamic, and precedes the development of postpartum disease.
   c. Accumulation of fat in the liver disturbs hepatic architecture and function, resulting in hypoglycemia and ketosis. There is also concurrent leukopenia, which may be related to the increased incidence of postpartum diseases seen with the condition.
4. Diagnostic plan
   a. Individual. Liver biopsy is the most reliable indicator of the condition; however, interpretation is difficult. Clinical pathology results are unreliable. Basing the diagnosis on clinical findings, history, and unresponsiveness to therapy often results in a diagnosis by default. This method is prone to error but is the diagnostic plan followed by clinicians.
   b. Herd. On a herd basis, overly fat cows with gestational ketosis and an increase in postpartum diseases leads to a presumptive diagnosis.
5. **Laboratory** tests
   a. Urinalysis. Urine ketone bodies may range from low to high.
   b. Serum liver enzyme studies. There may be a mild increase in aspartate aminotransferase (AST); however, this is nondiagnostic. Other liver enzymes may be elevated, but the relationship to disease is not invariable.
   c. CBC. There may be leukopenia, neutropenia, and lymphopenia.
   d. A liver biopsy is the most reliable and best correlated ancillary test. Fat content may be estimated by the specific gravity (SG) of the sample. Specimens for pathology often are available when there is a herd problem.
6. Therapeutic plan
   a. Standard therapy. Treat concurrent or clinical disease. Promote feed intake with palatable feeds, good quality hay, rumen transfaunation, and the provision of substrate for rumen microbes. Administer intravenous dextrose and oral propylene glycol.
   b. **Other** therapies that may be attempted in the individual include choline chloride at 25 g every 4 hours subcutaneously or orally, protamine-zinc insulin (200 IU intramuscularly every other day), oral niacin, and glucocorticoids (as in ketosis). Steroids should be used with caution in cases of animals with inflammatory disease.
7. Prognosis and prevention. The prognosis is poor for clinically affected animals. Prevention should center on avoiding obesity in cows. Monitoring body condition scores is helpful in this regard as is separation of cows into feeding groups based on performance and stage of gestation. Exercise is beneficial. Supplement the ration with oral niacin or nicotinic acid (6–12 g/animal/day) for 1–2 weeks prepartum and for 90–100 days postpartum.

H. Primary protein and energy malnutrition (starvation, emaciation)

This condition may be caused by a primary lack of feed or secondary to parasitism or a disease process that causes anorexia, an increase in metabolic rate, or both. This chapter considers primary protein and energy malnutrition relating to insufficient feed or improper nutrients.

1. **Patient** profile and history. **Feed** insufficiency most often occurs in pregnant or growing animals, and may be the result of poor husbandry or mismanagement. Poor-quality feed in either the roughage or grain component may cause malnutrition. It is often a herd or flock problem.
2. Clinical findings
   a. Individual
      (1) Affected animals may appear normal to the owner one day and recumbent the next. This is a common finding despite the chronic nature of the disease. Loss of body condition may be appreciated by palpation over the ribs and the spinous processes of the vertebrae. Loss of fat and muscle mass will be felt.
      (2) Animals present with an alert mental status despite recumbency. There is hypothermia with a normal to avid appetite. The GIT is usually hypomotile, and in ruminants, the rumen is firm. Diarrhea may be present terminally. Pregnancy usually continues, to the detriment of the dam.
   b. Herd
      (1) In cattle, there is delayed sexual maturity and infertility on a herd basis and signs of poor performance and emaciation. Beef cows that carry fetuses to term have low calf birth weights, decreased milk production, increased calf mortality, and decreased calf weaning weights. Dystocias may increase because of the small size of dams.
      (2) In sheep, clinical signs are similar to those in cattle with the added finding of decreased numbers of multiple births.

(3) In goats, the social order may result in malnutrition in submissive animals.
(4) Malnutrition adversely affects the immune system, therefore, the prevalence of diseases may increase. Terminally, there is hypoglycemia and bradycardia.

3. **Etiology** and pathogenesis. Insufficient feed may be associated with environmental factors (e.g., heavy snow cover, poor growing season, inclement weather).
   a. With general underfeeding, energy is usually the limiting factor but does not often occur independent of protein deficiency. Because of the composition of livestock rations, pure protein deficiency is less common in animals than in people. It can occur with home-mixed feeds where attention is not paid to protein and energy compositions.
   b. With limited nutrients, animal maintenance requirements (including growth of the fetus) receive priority. Requirements for growth, production, and fertility will not be met. Body stores of protein and energy are mobilized in the following order:
      (1) Glycogen—from limited stores
      (2) Lipid—as nonesterified FAs from body fat. (Incomplete oxidation of the FAs occurs, but glucose demand is moderate so that ketosis does not occur.)
      (3) Protein—as the major source for glucose to satisfy energy needs. (Catabolism is initiated by decreased insulin and increased corticosteroid secretion.)
   c. In advanced cases of starvation, rumen microbes become depleted (because of lack of substrate), as do digestive enzymes. This, coupled with hypoproteinemia and bowel edema, helps explain the rapid deterioration observed in these cases, the prolonged response to therapy, and the diarrhea that may occur. When adequate diets are offered to the animal with malnutrition, diarrhea is often an early response.

4. Diagnostic plan. The diagnosis is based on history and clinical and subjective findings. It is necessary to rule out other conditions and primary diseases that may be interfering with intake or utilization of feedstuffs. Necropsy findings are diagnostic (serous atrophy of fat).

5. **Laboratory** tests are often not reliable and are of little value in the individual animal. Hypoglycemia is a common finding but is not diagnostic.

6. Therapeutic plan. Treatment of the recumbent animal is often unrewarding.
   a. For animals not recumbent and with a good appetite, offer high-quality forages with gradual introduction to concentrate feeding. Force feeding may be helpful combined with transfaunation of microbes. Induction of parturition or cesarian section to relieve fetal demands for energy may be necessary.
   b. Supportive care for recumbent animals should be introduced (e.g., rolling, deep bedding, easy access to food and water). Therapy may be very expensive if total parenteral nutrition is employed.

7. Prevention. Discuss with the owner the provision of supplemental feeding for the herd or flock. High-quality roughage and natural protein supplements (e.g., soybean meal) should be provided. Good management practices, such as the provision of shelters, attention to animals (body condition scoring), adequate feeder space, group feeding, feed testing, and understanding cold weather needs, are essential.

I. Hypomagnesemic tetany

1. Patient profile and history. The manifestations and epidemiology of hypomagnesemia are variable. All types are usually seen sporadically in individual animals. This condition is seen in:
   a. Mature dairy cattle in early lactation and often on pasture (also called **lactation** tetany, grass staggers, grass tetany)
   b. Calves (age **2–4** months) fed exclusively whole milk or other diets low in magnesium
   c. Cattle or sheep grazing on young green cereal crops (also called wheat pasture poisoning)
   d. Beef cattle on poor pasture, during changeable, inclement weather (cold, rain, and wind)

2. Clinical findings
   a. Lactation tetany
      (1) Acute form. Animals may be found dead or with signs of a sudden onset of excitement, hyperesthesia, and frenzy. Cows fall into lateral recumbency in a rigid tetanic spasm, undergoing clonic/tonic convulsions with muscle fasciculations, opisthotonos, nystagmus, chewing movements, and snapping of the eyelids. These short, intense episodes are interspersed with quiet periods but may be precipitated again by noise or physical stimulation. There are very loud heart sounds. Vital signs are elevated, and the mortality rate is high because death often ensues before treatment can be administered.
      (2) Subacute form
         (a) There is a more gradual onset of signs for 2–3 days with anorexia, hyperesthesia, and some signs of cortical stimulation. Muscle fasciculations, an unsteady gait, trismus, spasmodic, pulsatile urination, and defecation are observed. Milk yield is decreased.
         (b) Spontaneous recovery may occur, or there may be progression to advanced clinical signs if the animal's condition is exacerbated by a precipitating event (e.g., physical stimulation). This condition is milder than the acute form, and treatment is often successful.
   b. Hypomagnesemic tetany of calves
      (1) Early signs are exaggerated movements, hyperesthesia, and a hyper alert attitude. There is opisthotonos, ataxia, and jerky limb movements. A backward carriage of the ears is a characteristic finding.
      (2) Signs progress to muscle fasciculations, spasticity, and convulsions. Tetany is not a finding of this condition, despite the name; however, the jaws are clenched tightly during clonic/tonic convulsions. Respirations cease during convulsions, and involuntary urination and defecation occurs. The heart rate is rapid, and heart sounds are very loud. Older calves often die, whereas younger calves may recover for short periods of time. Diarrhea may be a finding.
   c. Hypomagnesemic tetanies of beef calves, sheep, or cattle grazing cereal pastures. This condition has clinical signs similar to those of lactation tetany in dairy cattle.

3. Etiology and pathogenesis
   a. Etiology. Hypomagnesemia occurs with low dietary intake (because there are ***no*** readily mobilized magnesium reserves in the body), excessive **magnesium loss** (lactation, urinary loss), or both. Young calves absorb dietary magnesium very well, but this ability declines rapidly with age.
      (1) Low dietary intake may occur with poor pasture, cereal pasture, milk diet, or lush-growing forage (grasses). Lush pasture growth may occur in the spring, particularly with heavy potassium or nitrogen fertilization. High potassium content of plants may compete with magnesium absorption.
      (2) Excessive magnesium loss. Lactation is the main source of excessive magnesium loss, but magnesium is also lost through urine.
   b. Pathogenesis. Hypomagnesemia causes the clinical signs and may be precipitated by a sudden period of starvation (e.g., as with inclement weather). Hypocalcemia may be contributory and precede the development of clinical signs in chronically hypomagnesemic animals. Tetanic convulsions may result from **CNS** stimulation because of low magnesium levels in the cerebrospinal fluid (CSF).

4. Diagnostic plan. Diagnosis is based on clinical signs, response to therapy, serum magnesium levels (individual and herdmates), and serum calcium results.

5. **Laboratory** tests
   a. Serum magnesium levels are depressed (less than **0.2–0.4 mmol/L;** normal is 0.8–1.3 mmol/L). However, serum levels may be variable or higher during episodes of muscular contractions (tetany) because of the release of intracellular magnesium. Magnesium levels in herdmates should be measured and may be low (0.4–0.8 mmol/L).
   b. Serum calcium levels are often reduced (1.25–2 mmol/L).

c. Urinary magnesium levels are below normal (less than 0.4 mmol/L).
d. Pathology findings include low magnesium levels in the vitreous humor of the eye if specimens are collected within 48 hours and the animal has been kept cool. CSF magnesium levels (less than 0.5 mmol/L) are diagnostic if collected within 12 hours.

6. Differential diagnoses include CNS diseases (e.g., lead poisoning, rabies, nervous ketosis, polio in calves, tetanus, hypovitaminosis A).

7. Therapeutic plan
   a. Handle animals quietly before treatment. **Tranquilization** may be necessary.
   b. Treat animals with calcium and magnesium salts. A recommended treatment is 500 ml of a commercial calcium and magnesium preparation (50 ml for sheep and calves) administered slowly intravenously. This should be followed by 200 ml of 50% solution of magnesium sulfate (20 ml in preruminants or small ruminants) subcutaneously to maintain serum levels.
   c. Follow initial treatment with oral magnesium supplementation at the rate of 60 g magnesium oxide/day for 7 days for cows or 1–3 g/day for 6 weeks for calves and small ruminants.

8. Prevention
   a. Counsel owners regarding ways to provide increased levels of dietary magnesium (e.g., feeding of magnesium supplements to affected individuals and herdmates, spraying pastures with magnesium sulfate or other magnesium preparations, fertilizing pastures with magnesium products, spraying magnesium on hay). Conversion of pastures from grasses to legumes also increases forage levels of magnesium.
   b. To limit environmental and managemental risk factors for this condition, consider the provision of shelter, adequate nutrition, and supplementing calf diets with legume hay and grain.

II. METABOLIC DISORDERS OF HORSES

A. Hypocalcemic tetany of mares (lactation tetany, eclampsia, transit tetany)

1. Patient profile and history. There are two classic presentations of this condition:
   a. Lactation tetany, which is seen in heavy milking draft horses at approximately 10 days post foaling or 1–2 days post weaning
   b. Transit tetany, which is described in lactating or nonlactating animals transported long distances

2. Clinical findings. In both presentations, severely affected animals exhibit tetany and incoordination. Horses are apprehensive and sweating with dilated nostrils, tachypnea, and synchronous diaphragmatic flutter (SDF). Muscular fibrillations and a rapid, irregular pulse are observed. Affected horses are unable to eat, drink, or swallow, and it may not be possible to pass a nasogastric tube. Clinical signs advance to recumbency with tetanic convulsions.

3. Etiology and pathogenesis
   a. **Hypocalcemia** is thought to be responsible for the clinical signs, although either hypomagnesemia or hypermagnesemia may be seen in some cases. Hypocalcemia may be produced by heavy lactation, transport, hard physical work, or no apparent cause.
   b. **SDFs** are thought to be the result of diaphragmatic contractions synchronous with the heart beat and caused by changes in the excitation potential of the phrenic nerve because of electrolyte imbalances.

4. Diagnostic plan and laboratory tests. The diagnosis is made on the basis of clinical and subjective findings and confirmed by serum calcium levels. There is hypocalcemia (1–1.5 mmol/L). Serum magnesium values are variable, but hypomagnesemia may be seen with transit tetany (0.4 mmol/L).

5. Differential diagnoses include tetanus, laminitis, enteritis, and colic.

6. Therapeutic plan. Calcium solutions are administered intravenously (commercial preparations contain 8 g of calcium per 500 ml). Responses to therapy are gradual, lessening the signs of tetany and the voiding of large volumes of urine. The response is usually good in individual animals.

7. Prevention. Oral calcium supplementation or increasing dietary availability of calcium may be considered in heavily lactating mares.

B. Hyperkalemic periodic paralysis (HYPP)

1. Patient profile and history. HYPP is a genetic disease of Quarter horses, Appaloosas, American point horses, and Quarter horse crosses. Gene frequency is highest in one pedigree of these breeds (i.e., affected horses are all descendants of a single Quarter horse sire). Animals most frequently observed with the condition are well-muscled males age 2–3 years. The history and observed findings may indicate periods of prolonged recumbency or cutaneous abrasions.

2. Clinical findings
   a. Clinical attacks may be triggered by chilling, transportation, exhibitions at shows, and other stressors. In heterozygotes, the most common and earliest clinical sign is muscle fasciculations, followed by muscle spasms of the face, jaws, and legs. Weakness and recumbency follow. Death may ensue due to respiratory or cardiac failure, but this is rare. Recovery may take several minutes to several hours. Animals are normal between episodes.
   b. Many horses have increased respiratory rates during attacks and may show stridor if laryngeal or pharyngeal muscles are affected. Marked and persistent dyspnea may occur in homozygous foals. There are reports of HYPP attacks following anesthesia.

3. Etiology and pathogenesis. HYPP is similar to the human condition and has been transmitted as an autosomal dominant trait that is most likely from a single sire. The disorder is produced by failure of ion transport across the skeletal muscle cell membrane due to an abnormality in the sodium channel. In horses, this has been localized to a point mutation that changes an amino acid (e.g., phenylalanine, leucine) in the α-subunit of the sodium channel protein.
   a. **Defective** sodium channels remain open after membrane depolarization, allowing excessive inward sodium movement and heightened membrane depolarization. Simultaneously, normal sodium channels may be inactivated, preventing normal action potentials from developing. This creates muscular weakness.
   b. **Hyperkalemia** may be secondary to increased release of potassium as potassium channels open to depolarize the muscle membranes. Hyperkalemia also may occur if potassium is less able to enter the myocytes, resulting in serum accumulation.

4. Diagnostic plan and laboratory tests
   a. The condition may be diagnosed subjectively by clinical findings and objectively by elevated serum potassium levels. Serum potassium is usually elevated to between 6 mmol/L and 8 mmol/L during attacks. These elevations only persist for 1–2 hours. Serum should be separated from clotted samples as soon as possible to prevent red blood cell leakage of potassium into the serum.
   b. Definitive diagnosis is based on a gene probe for HYPP-type sodium channel DNA. The test is based on analysis of whole blood and is available commercially.
   c. A potassium chloride challenge test also has been used to diagnose the condition; however, this test is difficult to interpret and may be fatal in some horses. Potassium is administered orally at 0.1 g KCl per kg, and both clinical signs and blood potassium levels are monitored. If the horse shows signs of muscle fasciculations and hyperkalemia, the test is discontinued. If results are negative, the test

may be repeated up to four more times, increasing the administered potassium by 0.025 g KCl per kg every challenge up to a total potassium dose of 0.2 g/kg in adults or 0.15 g/kg in foals or weanlings.

d. Electromyography of suspect horses is practiced for immediate diagnostics and is quite reliable (90% reliability).

5. Differential diagnoses for presenting clinical signs include colic, trauma, and exertional rhabdomyolysis.

6. Therapeutic plan
   a. Emergency treatment consists of the intravenous administration of **5%** dextrose (2 ml to 6 ml/kg) together with sodium bicarbonate (1 mmol/kg to 2 mmol/kg) or **23%** calcium gluconate (0.2 ml/kg). Dextrose and bicarbonate will move potassium back into cells. Calcium counteracts the effects of hyperkalemia.
   b. Long-term therapy involves removing high-potassium feeds from the diet (e.g., protein supplements, bran, sweet feeds) and feeding a diet low in potassium (e.g., whole grains, grass hay). Medical therapy may be used, including acetazolamide (a potassium-wasting diuretic) at 2 to 3 mg/kg orally, 2 or 3 times a day. The dose may be decreased over time until the lowest effective dose is established.

7. Prevention. There is no cure for HYPP, which is a genetic disease and is inherited as an autosomal dominant trait. HYPP has been diagnosed more frequently during the last few years most likely because the carriers of the condition have been used heavily as sires. Because of the characteristics these carriers have (e.g., well-proportioned, heavily muscled appearance), the disease probably has been unknowingly propagated. Owners should be counseled about parentage identification and DNA gene probe testing. HYPP-positive horses should be removed from breeding use.

C. Hyperlipidemia (**hyperlipemia**) is discussed in Chapter 5 II A **4.**

III. METABOLIC DISORDERS OF NEONATES.

Neonatal hypoglycemia may occur in all neonates but is most common in newborn and weak lambs or piglets. This condition is seen in calves with diarrhea and may be a common cause for crushing deaths, which occur when sows crush piglets against the sides of farrowing crates. Crushing deaths are often accidental, but the incidence increases in piglets with hypoglycemia who are too weak to move out of the sow's way.

A. Clinical findings. Animals are shivering, dull, and anorexic. Affected animals have subnormal rectal temperatures, bradycardia, and soft heart sounds. Terminally, convulsions are followed by coma and death.

B Etiology and pathogenesis

1. In neonatal pigs, liver glycogen is rapidly depleted (within 12–24 hours) to maintain blood glucose. Therefore, because the neonate has little carcass fat and poor insulating capacity, the piglet is totally dependant on ingested milk as an energy source. Milk availability may be limited if there is disease in the sow (e.g., mastitis-metritis-agalactia), large litters and competition, sow hysteria, or diseases in the piglet (e.g., transmissible gastroenteritis). In piglets, the critical period lasts until 1 week of age, after which lack of intake only produces weight loss, not hypoglycemia.
2. In lambs, twinning, mismothering, and hypothermia all produce clinical hypoglycemia.
3. In calves, the predisposing factors are hypothermia, diarrhea, and improper or insufficient nutrition. The condition may occur in older calves receiving milk or milk replacer with little or no supplemental feed.

C. Diagnostic plan and laboratory tests. The diagnosis is based on clinical signs, subjective assessment, and response to therapy. A laboratory finding of hypoglycemia is confirmatory. Blood glucose findings below 2.2 mmol/L are diagnostic.

D. Differential diagnoses include diseases of the CNS. Gastrointestinal disease may be a concurrent and causative condition.

E. Therapeutic plan

1. Piglets. Treat with 15 ml of 20% dextrose every 6 hours. It may be necessary to provide a foster dam or an artificial diet. Correct the environmental temperature.
2. Lambs. Administer 10 ml/kg of 20% dextrose intraperitoneally. Rewarm via warm water baths.
3. Calves. Treat with 200 ml of 5% dextrose150 kg administered rapidly intravenously. Follow with 240 ml/50 kg/hr, and recheck glucose in 1 hour.

F. Prevention

1. Provide a warm environment for all neonates. Cross-foster offspring from multiple births or with poor milking or mothering dams. Treat concurrent disease in dams or offspring. Provide early colostrum to preruminants (force feed if necessary). Good o b servation is important
2. Do not withhold milk too long from diarrheic calves (maximum 48 hours), and feed small portions frequently. Increase nutrition for calves during cold weather (12%–14% body weight).

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. Which statement regarding parturient paresis (milk fever) of cattle is correct?

(1) It is caused by calving injuries.
(2) It is relatively rare in heifers.
(3) It produces a fever in stage III presentations.
(4) It causes the heart to sound loud and pounding.
(5) It is associated with diarrhea.

2. Which one of the following findings is consistent with parturient paresis in cattle?

(1) Hypomagnesemia is often a laboratory finding.
(2) Mortality is high despite treatment.
(3) Paralysis occurs because of high calcium concentration at neuromuscular junctions.
(4) Serum phosphorus may be below normal limits.
(5) There is a positive response to anti-inflammatory drugs.

3. The term "downer cow" refers to:

(1) animals remaining recumbent for 24 hours after initially being unable to rise.
(2) milk fever.
(3) cows that have hypophosphatemia.
(4) cows that are recumbent and ill due to systemic disease.
(5) low milk production at the peak period of lactation.

4. Which one of the following statements regarding postparturient hemoglobinuria is correct?

(1) It is a primary renal disorder.
(2) It results from increased red blood cell fragility.
(3) It is secondary to liver disease.
(4) It is usually widespread within a herd.
(5) There is a favorable prognosis in clinically affected animals.

5. Which statement regarding primary bovine ketosis is correct?

(1) It results from insufficient dietary protein.
(2) It is signaled by a rapid decline in milk yield.
(3) It may be seen in beef cattle with diets low in acetic acid.
(4) It is mainly a disease of pregnant heifers.
(5) It may be seen in high producing dairy cattle with insufficient energy intake.

6. Pregnancy toxemia of sheep is best described as:

(1) a condition of range sheep grazing lush spring pastures.
(2) a metabolic disease resulting from kidney failure.
(3) a condition of heavily pregnant ewes carrying multiple fetuses.
(4) responsive to vitamin D and glucose therapy.
(5) resulting from hypocalcemia and hypophosphatemia.

7. Fat cow syndrome in beef cattle and pregnancy toxemia in sheep are similar in that:

(1) animals may present with nervous signs.
(2) the conditions are contagious.
(3) the diseases occur in early gestation.
(4) treatment carries a favorable prognosis.
(5) prevention centers around vaccination programs.

8. Lactation tetany of mares is caused by:

(1) clostridial mastitis.
(2) trauma from foaling.
(3) metritis.
(4) hypophosphatemia.
(5) hypocalcemia.

9. Which one of the following statements regarding hyperkalemic periodic paralysis (HYPP) is correct? HYPP is:

(1) diagnosed by a gene probe for HYPP-type sodium channel DNA.
(2) an autosomal recessive condition of standard breeds in standard-bred crosses.
(3) treated with oral sodium chloride until the animal outgrows the condition.
(4) seen only in animals younger than 2 years of age.
(5) a disease of poor, unthrifty animals on high phosphorus (bran) diets.

10. Hyperlipidemia in mares is best described as:

(1) a result of a high-fat diet.
(2) a disease of fat mares that often develops in late pregnancy.
(3) a common, nonpathologic occurrence.
(4) a disease that occurs spontaneously at the time of heaviest lactation.
(5) secondary to vascular disease.

ANSWERS AND EXPLANATIONS

1. The answer is 2 [I A 3]. Parturient paresis in cattle is caused primarily by hypocalcemia due to calcium losses in milk. Therefore, it is relatively rare in heifers whose lactation demands are not as high as those of older, high-producing cows.

2. The answer is 4 [I A 2 a, 5 b]. Although the primary cause of parturient paresis is hypocalcemia, serum phosphorus levels are often concurrently below normal. Hypermagnesemia also may occur but not hypomagnesemia. Paralysis is caused by low calcium availability at the neuromuscular junction necessary for the release of the neurotransmitter acetylcholine. Treatment is often successful with intravenous and subcutaneous administration of commercially available calcium.

3. The answer is 1 [I B 1]. "Downer cows" may have had milk fever and have not responded fully to calcium therapy by rising, but they are usually no longer hypocalcemic or hypophosphatemic. These animals also may suffer from musculoskeletal problems but are not systemically ill.

4. The answer is 2 [I C 3]. Postparturient hemoglobinuria results from low serum phosphorus levels, which cause increased erythrocyte fragility. This hypophosphatemia may be the result of low intake coupled with lactation losses. Intravascular erythrolysis causes hemoglobinuria and may result in secondary renal damage. Although the hypophosphatemia may be seen on a herd level, it is a condition of individual animals at the peak of lactation. The prognosis is poor to guarded in clinically affected cows.

5. The answer is 5 [I D 3]. Primary bovine ketosis is a hypoglycemia and ketonemia resulting from high glucose losses through lactation combined with insufficient intake or availability. It presents clinically as a gradual decrease in appetite and milk production. Diets known to be precursors for this condition are those high in butyric or acetic acids.

6. The answer is 3 [I F 1]. Pregnancy toxemia of ewes usually occurs in intensively managed sheep carrying multiple fetuses. These are usually over-conditioned animals that may have suddenly faced a declining level of energy in the ration. Terminally, there is renal failure due to shock and dehydration caused by the primary disease. The condition is poorly responsive to any treatment, but the treatment of choice is immediate cesarian section with concurrent glucose therapy.

7. The answer is 1 [I F 2 a, G 2 b (2)]. Fat cow syndrome affects overly fat cows. As in sheep with pregnancy toxemia, beef cattle in late gestation may be affected by fat cow syndrome. This is a noncontagious disease of overly fat animals with an unfavorable prognosis. Prevention of both conditions centers around avoidance of overly fat animals in late gestation and eliminating the possibility of periods of anorexia or starvation.

8. The answer is 5 [II A 3 a]. Lactation tetany of mares is caused by hypocalcemia. This disorder is different than hypocalcemia in dairy cows because cows present with recumbency and flaccid paralysis. The other choices do not cause lactation tetany.

9. The answer is 1 [II B 4]. There is a molecular genetic test to identify the gene mutation. The condition is seen mainly in Quarter horses and is treated in clinical situations with bicarbonate and glucose administered intravenously to drive potassium intracellularly. It is a condition of well-muscled animals, and there is no age predilection. Animals have this condition for life but can often be maintained on low-potassium diets.

10. The answer is 2 [II C]. Fat mares in late pregnancy are most prone to hyperlipidemia. It often occurs in over-conditioned animals at times of decreased feed intake. It is uncommon in occurrence and secondary to stress or disease. It produces a vascular thrombosis and hepatic and renal failure.